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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/681,421	10/07/2003	Michael Neal Blackburn	P50438-1C2	7413

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EXAMINER	
DUFFY, PATRICIA ANN	
ART UNIT	**PAPER NUMBER**
1645	

DATE MAILED: 07/19/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/681,421	BLACKBURN ET AL.	
	Examiner	Art Unit	
	Patricia A. Duffy	1645	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *14 June 2006*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-20* is/are pending in the application.

4a) Of the above claim(s) *14-16* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-13 and 17-20* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *10-7-03* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *2003*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Applicants response filed 6-14-06en entered into the record. Claims 1-20 are pending.

Information Disclosure Statement

The information disclosure statement filed 10-7-03 The information disclosure statement filed 10-7-06 fails to comply with 37 CFR 1.98(a)(1), which requires the following: (1) the application number of the application in which the information disclosure statement is being submitted on each page of the list and (2) a column that provides a blank space next to each document to be considered, for the examiner's initials.

The information disclosure statement has been placed in the application file, but the information referred to therein has not been considered.

Election/Restriction

Applicant's election of Group I, claims 1-13 and 17-20 and Species A factor IX/IXa monoclonal antibody in the reply filed on 6-14-06 is acknowledged. Because applicant did not distinctly and specifically point out the supposed errors in the restriction requirement, the election has been treated as an election without traverse (MPEP § 818.03(a)).

The requirement is still deemed proper and is therefore made FINAL.

Claims 14-16 are withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected inventions, there being no allowable generic or linking claim. Applicant timely traversed the restriction (election) requirement in Paper No. 13.

Specification

The title of the invention is not descriptive of the claimed invention. A new title is required that is clearly indicative of the invention to which the claims are directed.

Claim Rejections - 35 U.S.C. § 112

Claims 1-13 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of inhibiting thrombosis in an animal comprising administering an dose of anti-Factor IX monoclonal antibody having self-limiting neutralizing activity effective to inhibit thrombosis in combination with a plasminogen activator, does not reasonably provide enablement for inhibiting thrombosis using any antibody that binds any coagulation factor. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The claims are drawn to the administration of any anti-coagulation factor monoclonal antibody having self-limiting neutralizing activity in combination with a plasminogen activator in order to inhibit thrombosis in an animal. The teachings of the specification are limited to the demonstration of an anti-Factor IX monoclonal antibody having self-limiting neutralizing activity alone or in combination with tPA in order to inhibit thrombosis in an animal. Neither the art, nor the specification teach that all other claimed anti-coagulation monoclonal antibodies have (a) self-limiting neutralizing activity and (b) have the ability to inhibit thrombosis as instantly claimed. The different coagulation factors perform different functions in different portions of the coagulation cascade. All coagulation factors are not equivalent. Compensation of one part of the cascade when the other is defective or has lower activity has been documented in the art. However, such compensation is particular to the Factors involved. Applicants admit at page 64 line 1-10, that in a model of thrombosis the effect "... is significantly dependent on which anti-coagulant was administered with the thrombolytic.". The art of record teaches fibrinolytic agents such as tPA, APSAC, urokinase, TNK-tPA have different dosing

regimens and have different effective doses. In view of the different effects on patency of the different fibrinolytic agents and in view of the lack of teachings of the specification, is not readily apparent that the teachings for the combination of anti-Factor IX monoclonal antibody in combination with tPA has broad applicability to all anti-coagulation factor monoclonal antibodies having self-limiting neutralizing activity and thrombolytic agents as are instantly claimed. From this experimental data, which is limited to just two compounds, the skilled artisan could not predict the effects of any and all of the other antibodies and plasminogen activators as contemplated would be effective as claimed because it is unknown and unpredictable how these other substances would react in the perturbation of the extremely complex coagulation cascade, part of which is shown as Figure 27-3 on page 660 of Goodman and Gilman's The Pharmacological Basis of Therapeutics. As can be seen from the figure, many pathways exist to the creation and disposition of many of the factors involved in coagulation, and many substances (such as the C1 esterase inhibitor) can alter the physiological processes at multiple, and not single, points along the cascade. Because a particular factor can have beneficial effects in terms of treating thrombosis due to its action at one point in the cascade, but detrimental effects at some other point along these pathways, the net sum clinical effect of any factor cannot be predicted by the skilled artisan. The complexity of the system can also be shown by the Bone reference (1992) on page 1384, Table I. Many coagulation factors increase or decrease during sepsis and coagulation, and although Bone (1992) reviews various therapies disclosed in Table 2 (page 1385), it is clear from his review that the treatment of coagulation or thrombosis due to sepsis is an unpredictable area of medicine and that all of these theoretically possible therapies need to undergo much experimentation in order to determine their efficacies, with the ultimate treatment of coagulation induced by sepsis probably resulting in the administration of a "cocktail" of agents (page 1388). Therefore, the skilled artisan would be forced to perform undue experimentation to determine if any of the recited anti-coagulation factor monoclonal

antibodies (classical or intrinsic pathways) in combination with a plasminogen activator without chemical or structural limitations, would cover a vast and ever growing list of definable, but structurally and functionally distinct compounds which would have unknown and unpredictable effects, the disclosure provides insufficient examples or guidance to enable the skilled artisan to use the vast majority of the substances encompassed by the claim limitations without forcing undue experimentation to determine their efficacy, with the exception of monoclonal antibody that binds Factor IX combined with tissue plasminogen activator (tPA; which is a specific chemical compound known in the art) as the second component. "Coagulation factors" is a general term used in the art to describe a very large grouping of physically and functionally distinct proteins, which act upon another large grouping of physically and functionally distinct protein and protein receptors to produce their effects. There are dozens of different coagulation factors now recognized in the art. These coagulation factors have different functions and do not regulate the same process point in the pathway of coagulation. Their plasma and tissue levels are also distinctly and independently regulated, with different feedback mechanisms, different receptor types, different target cells, etc. All of these variables are not predictive one from the other. The physical make-up of these coagulation factors are also different, and the substances that will influence their various functions (formation, liberation, binding) will also be different relative to the particular coagulation factor. Given the broad scope and distinct subject matter encompassed by the term "coagulation factor," and "plasminogen activator" the specification is merely an invitation to experiment. The same reasoning and grounds of rejection apply to the functionally defined, but not structurally or chemically defined compounds recited in the claims. The instant situation is analogous to that which was addressed in *In re Colianni*, 195 USPQ 150, CAFC, which states that in the absence of examples, a method relying on a "sufficient" amount of therapeutic agent must disclose what the "sufficient" amount is and conditions associated with such. In the absence of such, a disclosure does not meet the requirements of 35 U.S.C. 112 first

paragraph. Therefore, the teachings of a particular combination of species does not extrapolate to the genus now claimed, in the absence of evidence that the findings for tPA and monoclonal antibodies to a coagulation factor are generic to the subgenus of plasminogen activators and the teachings for anti Factor IX monoclonal antibody having self-limiting neutralizing activity for the genus of anti-coagulation factor antibodies.

Claims 17-20 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of reducing a required dose of tPA comprising administering an anti-Factor IX monoclonal antibody in combination with tPA, does not reasonably provide enablement for a method of reducing a required dose of any thrombolytic agent. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The claims are drawn to a method of reducing a required dose of a thrombolytic agent comprising administering an anti-Factor IX monoclonal antibody in combination with the thrombolytic agent. The teachings of the specification are limited to the demonstration that Anti-Factor IX monoclonal antibody lowers the required dose of the plasminogen activator, tPA, to restore patency in an art accepted model of reperfusion thrombosis. Applicants admit at page 64 line 1-10, that the "..incidence of reperfusion is significantly dependent on which anti-coagulant was administered with the thrombolytic.". The art of record teaches fibrinolytic agents such as tPA, APSAC, urokinase, TNK-tPA have different dosing regimens and have different effective doses. In view of the different effects on patency of the different fibrinolytic agents and in view of the teachings of the specification, is not readily apparent that the teachings for the combination of anti-Factor IX monoclonal antibody in combination with tPA has broad applicability to all thrombolytic agents as are instantly claimed. Therefore, the teachings of a particular species does not extrapolate to the genus now claimed, in the absence of

evidence that the findings for tPA are generic to the subgenus of plasminogen activators or the genus of fibrinolytic agents. From this experimental data, which is limited to just two compounds, the skilled artisan could not predict the effects of any and all of the other antibodies and plasminogen activators as contemplated would be effective as claimed because it is unknown and unpredictable how these other substances would react in the perturbation of the extremely complex coagulation cascade that results from sepsis, part of which is shown as Figure 27-3 on page 660 of Goodman and Gilman's "The Pharmacological Basis of Therapeutics". As can be seen from the figure, many pathways exist to the creation and disposition of many of the factors involved in coagulation, and many substances (such as the C1 esterase inhibitor) can alter the physiological processes at multiple, and not single, points along the cascade. Because a particular factor can have beneficial effects in terms of treating thrombosis due to its action at one point in the cascade, but detrimental effects at some other point along these pathways, the net sum clinical effect of any factor cannot be predicted by the skilled artisan. The complexity of the system can also be shown by the Bone reference (1992) on page 1384, Table I. Many coagulation factors increase or decrease during sepsis and coagulation, and although Bone (1992) reviews various therapies disclosed in Table 2 (page 1385), it is clear from his review that the treatment of coagulation is an unpredictable area of medicine and that all of these theoretically possible therapies need to undergo much experimentation in order to determine their efficacies, with the ultimate treatment of sepsis probably resulting in the administration of a "cocktail" of agents (page 1388). Therefore, the skilled artisan would be forced to perform undue experimentation to determine if any of the recited coagulation factor monoclonal antibodies (classical or intrinsic pathways) in combination with a plasminogen activator without chemical or structural limitations, would cover a vast and ever growing list of definable, but structurally and functionally distinct compounds which would have unknown and unpredictable effects, the disclosure provides insufficient examples or guidance to enable the skilled artisan to use the vast majority of the

substances encompassed by the claim limitations without forcing undue experimentation to determine their efficacy, with the exception of monoclonal antibody that binds Factor IX combined with tissue plasminogen activator (tPA; which is a specific chemical compound known in the art) as the second component. "Coagulation factors" is a general term used in the art to describe a very large grouping of physically and functionally distinct protein ligands, which act upon another large grouping of physically and functionally distinct protein and protein receptors to produce their effects. There are dozens of different coagulation factors now recognized in the art. These coagulation factors have different functions and do not regulate the same process point in the pathway of coagulation. Their plasma and tissue levels are also distinctly and independently regulated, with different feedback mechanisms, different receptor types, different target cells, etc. All of these variables are not predictive one from the other. The physical make-up of these coagulation factors are also different, and the substances that will influence their various functions (formation, liberation, binding) will also be different relative to the particular coagulation factor. Given the broad scope and distinct subject matter encompassed by the term "coagulation factor," and "plasminogen activator" the specification is merely an invitation to experiment. The same reasoning and grounds of rejection apply to the functionally defined, but not structurally or chemically defined compounds recited in the claims. The instant situation is analogous to that which was addressed in *In re Colianni*, 195 USPQ 150, CAFC, which states that in the absence of examples, a method relying on a "sufficient" amount of therapeutic agent must disclose what the "sufficient" amount is and conditions associated with such. In the absence of such, a disclosure does not meet the requirements of 35 U.S.C. 112 first paragraph. Therefore, in the absence of further guidance from Applicants and in view of the limited teachings of the specification, it would require undue experimentation to practice the breadth of the claimed invention.

Claims 1-13, 18, and 19 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

As to claim 1-13, the phrase "effective amount" renders the claim indefinite because it is not clear from the claim construction what effect is achieved by the amount.

As to claims 5, 6, 18 and 19, the claims are rendered indefinite from the use of the term "..the monoclonal antibody has the identifying characteristics of ..." because it is unclear what the identifying characteristics of the recited monoclonal antibodies are particularly claimed. This rejection may be obviated by reciting ".. the monoclonal antibody having all the identifying characteristics of...". Should applicants amend the claims as recited, a deposit for patent purposes will be required because, the particular antibodies will be required to determine if the antibodies have all the identifying characteristics as those particular hybridomas.

Claim Rejections - 35 U.S.C. § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made

absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103 (c) and potential 35 U.S.C. 102(f) or (g) prior art under 35 U.S.C. 103(a).

It is noted that the instantly claimed invention drawn to methods of inhibiting thrombosis in an animal comprising administering an effective dose of anti-coagulation factor monoclonal antibody having self-limiting neutralizing activity in combination with a plasminogen activator, lacks written description in the parent application 08/783,853. Should Applicants wish to contest this, then applicants are invited to point to the parent '853 application where written description support for the claimed subject matter can be found. The filing date for prior art purposes is therefore the filing date of parent application 09/346,487, filed 7-1-99.

Claims 1-2 and 7-11 are rejected under 35 U.S.C. 103(a) as being unpatentable over Kessler (Chest, 99(Suppl 4):9112S, April 1991) in view of Ruf et al (Thrombosis and Haemostasis, 66(5):529-533, 1991).

Kessler teaches that the goal of current modern treatment of cardiopulmonary disease is predicated on the goal of dissolving the offending clot to establish vascular patency and preventing rethrombosis using anti-coagulants combination of anti-coagulative agents with lytic agents (see page 97S, column 1 abstract). Kessler teaches that similar therapies have been employed in the treatment of coronary artery thrombosis, pulmonary embolism and peripheral arterial occlusion. Kessler also teaches that when anti-coagulants or anti-platelet-aggregating agents are used sequentially or concomitantly as adjunctives to thrombolytic therapy, they appear to enhance its efficacy. Kessler et al teach the combination of aspirin, heparin or coumarin in combination with thrombolytic agents such

as tPA, streptokinase, and urokinase to treat thrombolytic disease (see for example page 101S, column 2, second full paragraph, page 102S column 1 and page 105S, column 2, second full paragraph). Kessler et al teach that the extrinsic pathway of coagulation activated by tissue factor or tissue thromboplastin can be monitored by the prothrombin time determination. Kessler teaches that tissue factor can be activated by factor IXa (see page 103S, column 2, last paragraph). Kessler differs by not teaching the administration of a monoclonal antibody against a coagulation factor to inhibit coagulation or rethrombosis.

Ruf et al teach anti-tissue factor monoclonal antibodies that inhibit TF-VIIa complex are potent anticoagulants. Ruf et al teach that the monoclonal antibody introduces a therapeutic principle for rapid arrest of inappropriate triggering of coagulation by tissue factor (page 529, summary). Ruf et al teach that these antibodies appear to possess therapeutic potential as exemplified by their capacity to provide prophylactic protection in the lethal *E. coli* septic shock model in baboons (page 529, paragraph bridging columns 1-2). Ruf et al teaches that the various specific and rapid inhibitors of TF-VIIa function in the extrinsic pathway have use in as tools for the study of the extrinsic pathway as well as therapeutic intervention in various states of disease (page 532, column 2).

It would have been prima facie obvious to one having ordinary skill in the art at the time the invention was made to substitute the coagulation inhibiting monoclonal antibody of Ruf et al for any one aspirin, heparin or coumarin in the treatment combination with thrombolytic agents such as tPA, streptokinase, and urokinase to treat thrombolytic disease because Kessler et al teach goal of current modern treatment of cardiopulmonary disease is predicated on the goal of dissolving the offending clot to establish vascular patency and preventing rethrombosis using anti-coagulants combination of anti-coagulative agents with lytic agents (see page 97S, column 1 abstract) and because Kessler also teaches that when anti-coagulants or anti-platelet-aggregating agents are used

sequentially or concomitantly as adjunctives to thrombolytic therapy, they appear to enhance its efficacy. The substitution of one anticoagulant for another in the combination therapy for treatment of any thrombolytic disease is *prima facie* obvious.

Claims 1-13 and 17-20 are rejected under 35 U.S.C. 103(a) as being unpatentable over Kessler (Chest, 99(Suppl 4):9112S, April 1991) and Ruf et al (Thrombosis and Haemostasis, 66(5):529-533, 1991) as applied to claims 1-2 and 7-11 above and further in view of Sheth et al (Blood et al., 92(10 Suppl. 1, Part 1-2), p 362A, November 15, 1998).

The combination of Kessler (Chest, 99(Suppl 4):9112S, April 1991) and Ruf et al (Thrombosis and Haemostasis, 66(5):529-533, 1991) is set forth supra. The combination differs by not using a monoclonal antibody that binds Factor IX or Factor IXa.

Sheth et al teach that administration of a humanized monoclonal antibody SB 249417
to healthy volunteers. Sheth et al teach that antibody binding inhibits activation of factor IX and also blocks the activity of Factor Xa on FX. Sheth et al teach the anticoagulant activity of SB 249417 was demonstrated by dose dependent increases in aPTT and ACT. Seth et al teach that SB 249417 may provide a new long acting anti-thrombolytic agent.

It would have been *prima facie* obvious to one having ordinary skill in the art at the time the invention was made to substitute the humanized monoclonal antibody of Seth et al for the anticoagulant monoclonal antibody of Ruf et al in the method of treatment of thrombosis as combined *supra* because Seth et al teaches that the humanized monoclonal antibody increases aPTT (i.e. inhibiting coagulation) and Kessler teaches that the extrinsic pathway of coagulation is monitored by the prothrombin time determination and that factor IX/IXa (see page 103S, column 2, last paragraph) participates in the coagulation by the extrinsic pathway. One would have been motivated to substitute the antibody of Seth et al in the method as combined because Seth et al teaches that the humanized

antibody inhibits coagulation as assessed by a prolongation of aPTT and ACT and Kessler et al teaches combination of agents enhance the efficacy of lytic agents.

Status of Claims

No claims are allowed. Claims 1-13 and 17-20 stand rejected. Claims 14-16 are withdrawn from consideration.

Citation of Pertinent Art

The prior art made of record and not relied upon is considered pertinent to applicant's disclosure.

Bajaj et al," Human Factor IX and Factor IXa", in Methods in Enzymology, Volume 222, Part A, pages 96-128 and 177, 1993 is cited to teach the structure and function of coagulation Factor IX and its function in all pathways of coagulation.

Conclusion

This is a continuation of applicant's earlier Application No. 09/965,099. All claims are drawn to the same invention claimed in the earlier application and could have been finally rejected on the grounds and art of record in the next Office action if they had been entered in the earlier application. Accordingly, **THIS ACTION IS MADE FINAL** even though it is a first action in this case. See MPEP § 706.07(b). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the

advisory action. In no, however, event will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Patricia A. Duffy whose telephone number is 571-272-0855. The examiner can normally be reached on M-Th 7:30 pm - 6:00 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Smith Lynette can be reached on 571-272-0864. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Patricia A. Duffy

Primary Examiner

Art Unit 1645